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Strategies for managing oxygenation in obese patients undergoing laparoscopic surgery

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Abstract

The worldwide trend toward increasing body mass index (BMI) has caused the anesthetic management of overweight, obese, and severely obese patients to become common. The increase in oxygen demand coupled with the anatomic and physiologic changes associated with excess adipose tissue make maintenance of oxygenation a major challenge during induction, maintenance and recovery from general anesthesia. It is crucial for anesthesiologists, surgeons and perioperative healthcare providers alike to have a thorough understanding of the impact of airway management and mechanical ventilation on the respiratory care of the obese in the immediate perioperative setting. In this manuscript we aim to discuss the consequences of obesity, particularly abdominal obesity, on respiratory physiology and provide suggestions on intraoperative ventilatory strategies to maintain oxygenation in the severely obese patient undergoing pneumoperitoneum.

Keywords

Body mass index (BMI); Pneumoperitoneum; Obesity; Perioperative management; Airway management; Ventilation; Oxygenation; General anesthesia

Case description

An 18-year-old man (height = 175 cm, weight = 243 kg, BMI = 79.3 kg/m^2) with a history of obesity since the age of 6 years was scheduled for laparoscopic Roux-en-Y gastric bypass. His medical history included depression, mild asthma, active smoking, hypertension, gastroesophageal reflux, and severe obstructive sleep apnea (OSA) with an apnea-hypopnea index of 27.6 events/hr. He used nighttime continuous positive airway pressure (CPAP) and the only medication he was regularly taking was esomeprazole.

Physical examination on the day of surgery revealed a blood pressure of 172/79 mm Hg, a heart rate of 84 beats/min, and a room air peripheral oxygen saturation (SpO_2) = 98%. His obesity was predominantly centrally distributed.

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After the initiation of standard monitoring, the patient was placed in a 30-degree, reverse Trendelenburg position and preoxygenated with 100% oxygen until the end-tidal oxygen concentration reached 85%. Preoxygenation was facilitated by intermittently asking the patient to take deep breaths. Rapid sequence induction with propofol (2.5 mg/kg of lean weight) and succinylcholine (0.6 mg/kg of total weight) followed and direct laryngoscopy with a MAC 3 blade revealed only a large epiglottis. A subsequent laryngoscopy with a Glidescope 4 blade (Verathon Medical ULC, Burnaby, BC, Canada) yielded a view of the glottis and a significant amount of redundant oropharyngeal tissue. This view permitted the insertion of a gum-elastic bougie between the vocal cords. A 7.5 mm (internal diameter) cuffed endotracheal tube was advanced over the bougie into the trachea. Upon completion of intubation, the SpO_2 had decreased from 100% to 94%. Once correct endotracheal position was confirmed, 3 recruitment maneuvers (35 cm H_2O for 10 s each) were delivered with restoration of the SpO_2 to 100%.

Mechanical ventilation was initially set to volume control with a tidal volume (VT) of 500 mL, a respiratory rate (RR) of 16, an inspiratory-to-expiratory (I:E) ratio of 1:2, a positive end-expiratory pressure (PEEP) of 8 cmH_2O and a fraction of inspired oxygen (FiO_2) of 50%. The respiratory frequency was subsequently adjusted to maintain an end-tidal carbon dioxide partial pressure ($ETCO_2$) between 30 and 40 mmHg. The PEEP was set before leveling the operating room table.

Anesthesia was maintained with sevoflurane (minimum alveolar concentration = 1.0), morphine sulfate (total dose of 8 mg), cisatracurium .05 $mg•kg^{-1}•hr^{-1}$ and ketamine infusion of .25 $mg•kg^{-1}•hr^{-1}$ using 100 kg as the infusion weight [1]. Neuromuscular blockade was monitored at the adductor pollicis muscle with a train-of-four ratio (TOFR) with acceleromyography using a TOF-Watch®SX portable device (Organon, Dublin, Ireland). Core body temperature was maintained at >36°C with a forced-air warming blanket. The infusions were discontinued approximately one hour before the end of surgery.

The patient's respiratory compliance was 31 mL/cmH_2O while in the supine position. It decreased to 22 mL/cm H_2O after the establishment of carboperitoneum to an intraabdominal pressure of 16 mmHg. Upon resumption of the reverse Trendelenburg position, the compliance increased to 25 mL/cm H_2O. Over time, the SpO_2 decreased from 100% to 93%. Blood pressure, capnography waveform, heart rate, and rhythm remained stable. Clear, distant, bilateral breath sounds were auscultated. The ventilation mode was changed to pressure control to try to increase the mean alveolar oxygen tension, but there was no improvement in the SpO_2. Three recruitment maneuvers (35 cm H_2O for 10 s each) were delivered resulting in an increase in SpO_2 to 98%.

The surgical procedure was completed in 2.5 hours with minimal blood loss. After the release of the carboperitoneum and resumption of the reverse Trendelenburg position the VT was noted to be 970 mL, necessitating an adjustment in the pressure control settings. Before emergence, the acceleromyography-measured TOFR was >100%, thus no reversal agents were administered. Once awake the patient was extubated in the reverse Trendelenburg position and transported to the postanesthesia care unit (PACU). He was maintained in the sitting position with supplemental oxygen provided via face mask. Forty-five minutes after

his arrival, his Alderete score was 9. He was transitioned to 2 L/min oxygen via nasal cannula and directed to use incentive spirometry every hour. The patient was observed in the PACU for an additional 2 hours until his ward room became available. At the time of transfer, his SpO_2 on 2 L/min oxygen via nasal cannula was 98%.

Discussion

Obesity and respiration: Beyond size alone

Obesity affects several aspects of respiratory function. Increasing weight has been consistently linked to a decline in expiratory reserve volume (ERV) [2] and functional residual capacity (FRC) [3]. Jones et al. reported a reduction in ERV from 118% to 55% of predicted in seated patients whose BMIs ranged from 20 to >40 kg/m^2. FRC fell from 112% to 84% of predicted when the BMI increased from 20 to 30 kg/m^2. The FRC continued to decline with increasing BMI such that at a BMI of 45 kg/m^2, FRC fell below the “lower limit of normal” [4]. Total lung capacity (TLC) as well as the ratio of forced expiratory volume in 1 second (FEV_1) to forced vital capacity (FVC) tend to be well maintained in simple to moderate obesity but decrease as obesity becomes severe [5–7].

The typical pattern of respiration in the obese is characterized by a faster rate with smaller tidal volumes, compared with an individual of normal BMI. This deviation is consistent with a restrictive pattern of ventilation [8]. Changes in respiratory mechanics are driven by the increased load on the chest wall and the associated reduction in FRC. This chest wall load produces a shift of the normal chest wall pressure-volume curve to the right resulting in increased pressures to produce a similar volume change.

The distribution of excess adipose tissue is a critical determinant of the degree of impairment in pulmonary function. Compared with a more peripheral distribution of fat (pear-shaped, gluteofemoral, or gynecoid), excess central adiposity (visceral, apple-shaped, abdominal, or android) restricts diaphragmatic excursions while higher chest wall adiposity is correlated with lower lung volumes [5,8]. Measurements of regional distribution of excess fat, such as waist circumference or waist-to-hip ratio, may better correlate with pulmonary function impairment than BMI [9].

Centrally deposited fat may also adversely affect pulmonary function through its endocrine properties. Adipocytes are active in the production of adipocytokines (or adipokines), cell-signaling proteins known to mediate several obesity-related conditions such as insulin resistance, hypercoagulability and hypertension [10]. One such adipokine, leptin, is an anorexogenic agent produced by adipose tissue which binds to hypothalamic receptors to balance food intake with energy expenditure. Obesity is characterized by leptin resistance despite plasma levels proportional to the degree of body fat [11]. Elevated plasma levels of leptin have been negatively correlated with FEV_1 [12,13]. Interestingly, leptin receptors as well as low amounts of leptin production have been identified in bronchial tissue [14]. Another adipocyte-specific protein potentially affecting the pulmonary system is adiponectin. With potent antiinflammatory properties, its circulating levels have been found to be lower in obese humans compared to lean, particularly in those with higher amount of

visceral fat [15]. Lower levels of circulating adiponectin have been associated with lower FVC and FEV_1 [16].

Long-term exposure to proinflammatory adipokines coupled with injury presumably incurred by repeated opening / closing of small airways may lead to remodeling of airway structures and increased airway resistance [17] consistent with the finding of airway narrowing with increasing weight in young obese men [18]. In this study group, the adverse effect on airway diameter extended beyond that expected based on weight alone, suggesting that a chronic state of diminished FRC, as well as altered levels of adipokines, can potentially alter airway structure and function [18]. Additionally, in patients with OSA, airway caliber can be further compromised by an increase in the volume of surrounding soft tissue [19], alterations of neural control of pharyngeal dilator muscles [20,21], as well as changes in control of ventilation [22], increasing the probability of airway collapsibility during induction as well as during recovery from general anesthesia.

Oxygenation before intubation: Airway management in the obese

A one-year audit of airway complications during anesthesia, intensive care and emergency departments in the United Kingdom (NAP4) revealed an association between BMI >30 kg/m^2 and airway problems. Potential causative factors included poor airway assessment, inappropriate selection of airway management devices (e.g., use of supraglottic airway device instead of an endotracheal tube) and failure to anticipate and plan for problems [23,24]. Although no single finding on physical exam can accurately forecast difficulties with airway management, the coexistence of several features, including obesity, has been linked to difficulties with mask ventilation [25]. In a recent review of approximately 177, 000 general anesthetics, Kheterpal et al. found that independent predictors of both difficult mask ventilation and difficult laryngoscopy included BMI ≥30, age ≥46 years, male sex, crowded oropharynx, neck mass, radiation, large neck circumference, limited thyromental distance, sleep apnea, presence of teeth, beard, and limitations of cervical spine mobility and jaw protrusion [26].

In procedures necessitating a secure airway, a crucial decision is whether to intubate the trachea with the patient awake or asleep. A reassuring airway exam, particularly in the absence of prior history of airway difficulties, is likely to be followed by intubation while asleep. The decision to proceed to an awake-look or awake intubation hinges on a thoughtful evaluation of whether or not one can maintain oxygenation once the patient ceases to breathe. As even a seemingly easy airway can be met with difficulties, it is important to have well-rehearsed alternative plans. Video laryngoscopy (VL) offers an option either as a primary intubating technique or rescue technique when direct laryngoscopy fails [27]. VL provides a view of the glottic opening as seen from the laryngoscope blade, in contrast to the view provided by traditional laryngoscopes. It may offer a clear view when otherwise no view is available. A limitation of this technology is that a good view does not necessarily guarantee intubation success; sometimes adjuncts such as a gum-elastic bougie may be needed to successfully guide the endotracheal tube into the trachea [28,29]. Furthermore, VL may not be appropriate for patients with limited mouth opening or large upper airway or pharyngeal lesions. In these cases, fiberoptic intubation (FOI) remains the gold standard for

intubating the trachea. Provider decision against securing the airway with an awake fiberoptic intubation was cited in the NAP4 report as a potential contributor to adverse airway outcomes [30]. Isono admonishes that: “Awake intubation should be considered when any element of the triple airway maneuver, including mandible advancement, neck extension and mouth opening, is disturbed in obese patients with severe OSA [31].” Though they lack protection against aspiration of gastric contents, supraglottic airway devices — including the laryngeal mask airway (LMA) — can also be used as a rescue technique for ventilation and intubation [32,33]. No specific device can restore oxygenation, but sound planning, clear communication and skillful use of available resources can facilitate navigation through a difficult airway scenario.

The patient’s position is an important determinant of perioperative FRC and, thus, the nonhypoxic apnea period. The reduction of FRC after induction of general anesthesia is greater in the obese than in the lean subject, particularly in the supine position [34]. Reduction of the abdominal load to the chest by the head-up or reverse Trendelenburg position (Fig. 1) should be performed whenever feasible as it confers improved patient comfort during spontaneous ventilation (e.g., preoxygenation) [35], a longer nonhypoxic apnea period [36], increased cross-sectional area at both retropalatal and retroglossal airway [37], and possibly, an improved view during direct laryngoscopy [38]. A disadvantage of this position is the potential for a decrease in blood pressure, especially if the patient is hypovolemic. Additionally, use of blankets under the upper body and occiput (“ramped position”) may facilitate neck flexion and extension at the atlanto-occipital joint to optimize the view at laryngoscopy. Application of CPAP and pressure support ventilation (PSV) during preoxygenation has been shown to achieve target preoxygenation more reliably and quickly [39,40] and to increase the postintubation partial pressure of oxygen in arterial blood (PaO_2) compared with tidal breathing in obese adults [41].

Oxygenation after intubation: Mechanical ventilation in the obese

After intubation, the combination of the patient’s physiologic dysfunction, surgical load such as Trendelenburg position, CO_2 loading, and pneumoperitoneum can compromise the patient’s homeostasis. Mechanical ventilation settings should be set to achieve a balance between optimizing gas exchange and minimizing lung injury and systemic (e.g., hemodynamic, neurologic) compromise. This implies minimization of barotrauma, volutrauma [42], low-volume lung injury [43], and biotrauma [44]. A number of studies and clinical practices have addressed the optimization of gas exchange during mechanical ventilation of the obese [45,46]. However, there are limited randomized prospective studies to determine which of the proposed ventilatory strategies are conducive to minimal lung injury.

Despite the fact that pressure control ventilation has been advanced in small studies as allowing for better mean alveolar pressure and improved gas exchange in the presence of lower peak inspiratory pressures, there is no evidence of its absolute benefit over volume control. When volume control is chosen, the tidal volume (VT) should be based on the patient’s predicted lung volumes to achieve appropriate ventilation while minimizing lung strain. Importantly, studies have repored that lung volumes depend on gender and height,

with no additional information added by weight [47,48]. Thus, use of the patient's weight to set VT in the obese risks overestimation of VT, and has been associated with worse outcomes [46]. A practical approach to individualize the patient' s VT is to compute it as proportional to the patient' s predicted weight (Table 1).

Respiratory rate (RR) is adjusted to normocapnia through its titration to a target $ETCO_2$ range of 30–40 mmHg; the goal is to maintain the patient's preoperative stable acid-base status. Of note, the gradient between $ETCO_2$ and the arterial partial pressure of carbon dioxide ($PaCO_2$) may be greater in obese patients than normal values of 2–5 mmHg. In obesity hypoventilation syndrome, a condition characterized by a higher baseline $PaCO_2$, the targeted $ETCO_2$ should reflect this higher $PaCO_2$ value. An arterial line should be used to more accurately guide the optimization of mechanical ventilation in advanced disease states and during major surgery.

Oxygen should be used as a medication to treat hypoxemia. The FiO_2 should be titrated to maintain normoxia, taking into consideration the patient's baseline values [49,50]. The determination of the optimal fraction of inspired oxygen for any particular patient is multifactorial. Most studies investigate a single outcome in isolation. Recommendations based on those outcomes are often contradictory: use lower FiO_2 to limit resorption atelectasis and oxidative stress to the lung [51], use higher FiO_2 to prevent surgical site infection [52] and post-operative nausea and vomiting [53]. The decision on which FiO_2 to use should reflect the balance of outcomes most pertinent to the patient. In the absence of an absolute need to restrict FiO_2, a range between 40–80% in the obese patient has been recommended [54].

Intraoperative surgical loads (CO_2 insufflation, patient position and abdominal pressure) [55] combined with the compromised respiratory mechanics and increased metabolic demands of the obese can impede delivery of minute ventilation sufficient to maintain the patient's preoperative acid-base status. Permissive hypercapnia is frequently used in these cases [56]. Specifically in the obese, Hager et al. found that mild hypercapnia ($ETCO_2$ 50 mmHg) improved tissue oxygenation without hemodynamic compromise [57]. Importantly, permissive hypercapnia is contraindicated in the presence of intracranial hypertension, pulmonary hypertension, significant right ventricular dysfunction, and significant atrial and ventricular arrhythmias. If intraoperative permissive hypercapnia is necessary, once surgical load is removed, ventilation should be aimed to return the patient' s baseline acid-base status before extubation.

Inspiratory-to-expiratory (I:E) ratio should be adjusted to the patient' s physiology. Compared with an individual of normal BMI, the typical pathophysiologic pattern of respiration in obese individuals is restrictive [8,58]. Settings ranging from 1:1 (restrictive physiology) to 1:2 (normal physiology) are acceptable. However, as a substantial fraction of chronic obstructive pulmonary disease (COPD) patients are obese, longer expiratory times (e.g., 1:3- obstructive physiology) may be required. The expiratory flow curve available in current anesthesia machines allows for direct determination of expiratory times.

PEEP is set to prevent lung derecruitment, which is important for oxygenation [59] and, potentially, lung protection. Initial settings at 6–12 cmH_2O are appropriate in the obese [45]. In the absence of other causes, persistent hypoxemia could suggest that even larger values may be required. In critical cases such as when high airway pressures are needed to maintain ventilation, measurement of esophageal pressure as an indirect estimate of pleural pressure (Ppleu) could be considered to support the determination of PEEP [45,60]. Higher values of PEEP should be used with meticulous attention to hemodynamics. Optimization of cardiac preload and afterload are important before substantial increases in PEEP. Stepwise increase of PEEP - in contrast to abrupt changes- can also help to minimize hemodynamic compromise. Because the pressure required to recruit collapsed lungs is larger than pressure needed to prevent collapse, it is desirable to apply the target PEEP after an RM [54,61] and before further lung derecruitment is expected (e.g., during reverse Trendelenburg, before a change in posture to supine or Trendelenburg).

Having determined the preceding settings, the plateau pressure (Pplat) needs to be checked. The limit of 26–30 cm H_2O has been traditionally regarded as a safe range in patients with adult respiratory distress syndrome (ARDS). The determination of an allowable Pplat in the obese is challenging because their body habitus combined with the surgical interventions may produce pleural pressures significantly larger than those present in lean patients. Because the pressure effectively deforming the lung is the transpulmonary pressure (Pplat-Ppleu)—not merely the Pplat—larger Pplat may still represent safe lung stretch in the obese [45,62]. Measurement of esophageal pressure to estimate Ppleu could support the choice of PEEP and allow for the computation of the transpulmonary pressures, allowing for a more physiologically founded choice of settings [45,62]. Tolerated values for peak inspiratory pressure (PIP) will be a few centimeters of water above the tolerated Pplat. The presence of risk factors for pneumothorax (e.g., known emphysema or bullae) and body positions resulting in larger gradients of transpulmonary pressures should be taken into account during the determination of allowable Pplat and PIP.

When pressure control is chosen as the mode of ventilation, the set inflation pressure above ambient should follow the rules given for the plateau pressure above.

Oxygenation after intubation: Body position and pneumoperitoneum

A decline in both chest wall and lung compliances with increasing BMI has been documented in anesthetized patients [34]. The decrease in lung compliance mainly reflects microatelectasis as lung volume is reduced [58]. Sprung et al. were unable to show a significant change in the measured respiratory compliance, resistance or oxygenation with postural changes, but showed a marked decrease in compliance and resistance with the institution of pneumoperitoneum to 20 mmHg. Surprisingly, little change in oxygenation was observed [55]. In contrast, Valenza et al. showed an improvement in oxygenation by the combination of placing the patient in the beach-chair position and adding PEEP, whereas they were unable to show an improvement by either intervention alone [63].

Recruitment maneuver (RM) techniques shown beneficial in the presence of hypoxemia and presumed atelectasis have included 30–40 cmH_2O for 30–40 seconds and 55 cmH_2O for 10

seconds [61,64]. RMs should be considered when an increase in PEEP is deemed to be necessary, and after maneuvers associated with lung derecruitment such as anesthesia induction and intubation, endotracheal tube disconnection, and increase in abdominal pressure. Accordingly, it is likely beneficial to establish lung recruitment before unfavorable changes in position such as Trendelenburg. In the severely obese, the combination of RM and PEEP is superior to each maneuver in isolation in reducing lung atelectasis and improving oxygenation after induction of general anesthesia [64].

Oxygenation after extubation

Obesity is a risk factor for airway complications. It is important to recognize that death and severe brain damage are more often associated with extubation or the recovery period than induction of general anesthesia [65]. In the NAP4, almost one third of all adverse events associated with anesthesia occurred at the end of the anesthetic or the recovery period [30].

To optimize pulmonary mechanics and access to the airway, extubation of the trachea in the obese should take place in the same position used for intubation (Fig. 1) upon verification of full reversal of neuromuscular blockade (e.g., TOFR ≥.9). Extubation should ensue when the patient is fully awake to minimize the possibility of airway obstruction. Immediately after extubation, the synchrony of thoracoabdominal movements should be assessed as paradoxical breathing may indicate pharyngeal obstruction. Common culprits of extubation failure in the obese include airway obstruction, bronchospasm and residual drug effects (e.g., muscle relaxants, opioids) [66]. Prompt recognition of extubation failure and, if necessary, reintubation are key to averting injury and death. In its recently published guidelines, the Difficult Airway Society advocates a deliberate, thoughtful plan for removal of the endotracheal tube [67]. It is imperative to have a plan to immediately regain control of the airway in the event that adequate ventilation is not achieved after extubation. For patients who were difficult to intubate, extubating over a tube-exchanger may be prudent should reintubation be required.

Utilization of CPAP has been purported to lessen the consequences of postoperative atelectasis, a frequent occurrence in the postanesthetic course of the obese patient. Although a recent Cochrane review concludes that there is no evidence that CPAP reduces mortality, it states that there may be a benefit in reducing atelectasis, preventing pneumonia and reducing the risk of reintubation [68]. The review underscores the need for further studies to confirm CPAP's benefit in the postoperative period. Interestingly, Gupta et al. noticed fewer serious postoperative complications (e.g., cardiac events) in OSA patients treated with preoperative CPAP than in those who did not receive preoperative CPAP [69]. In a recent study, OSA patients treated with CPAP for a 12-week period showed a reduction in mean arterial pressure whereas those who received nocturnal supplemental oxygen or education did not [70]. Despite the lack of solid data on the pre- and postoperative benefits of CPAP, recent practice guidelines from the American Society of Anesthesiologists [71] recommend that the "perioperative initiation of CPAP should be considered, particularly if OSA is severe." In the absence of contraindications, CPAP or noninvasive positive pressure ventilation (NIPPV) should be continuously administered postoperatively to patients who were using these modalities [71].

Conclusions

Obesity is a condition which affects all organs. Understanding its effect on the respiratory system is paramount to the maintenance of oxygenation in the immediate perioperative period. As periods of apnea are quickly followed by desaturation, preparation for airway management begins with a comprehensive airway assessment to help estimate the likelihood of a difficult intubation. To maximize available oxygen reserves during apnea, a period of preoxygenation aiming for an exhaled oxygen concentration of at least ≥85% should precede induction of general anesthesia. Unless contraindicated, preoxygenation, induction and emergence from general anesthesia should be conducted in the head elevated position. This posture allows gravity to assist in the downward displacement of the diaphragm, improving lung / chest wall expansion.

Mechanical ventilation of the obese can be one of the most challenging tasks faced by anesthesiologists. We have suggested parameters that seek to maximize lung protection while providing adequate gas-exchange. As it is easier to prevent atelectasis than to reverse it, it is advisable to add PEEP right after the trachea is intubated. The combination of PEEP and RM can be a powerful intraoperative tool to mitigate hypoxemia and optimize ventilation. Further work is needed to determine the long-term impact of these maneuvers.

The impact of obesity-related inflammatory mediators on pulmonary and airway structure and function is a potentially fruitful field of study. Further research is necessary to elucidate whether the relationship is causal (e.g., higher leptin levels cause a decrease in FEV_1) or not (higher levels of inflammatory mediators coexist with the obesity-related respiratory changes).

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Conflicts of interest

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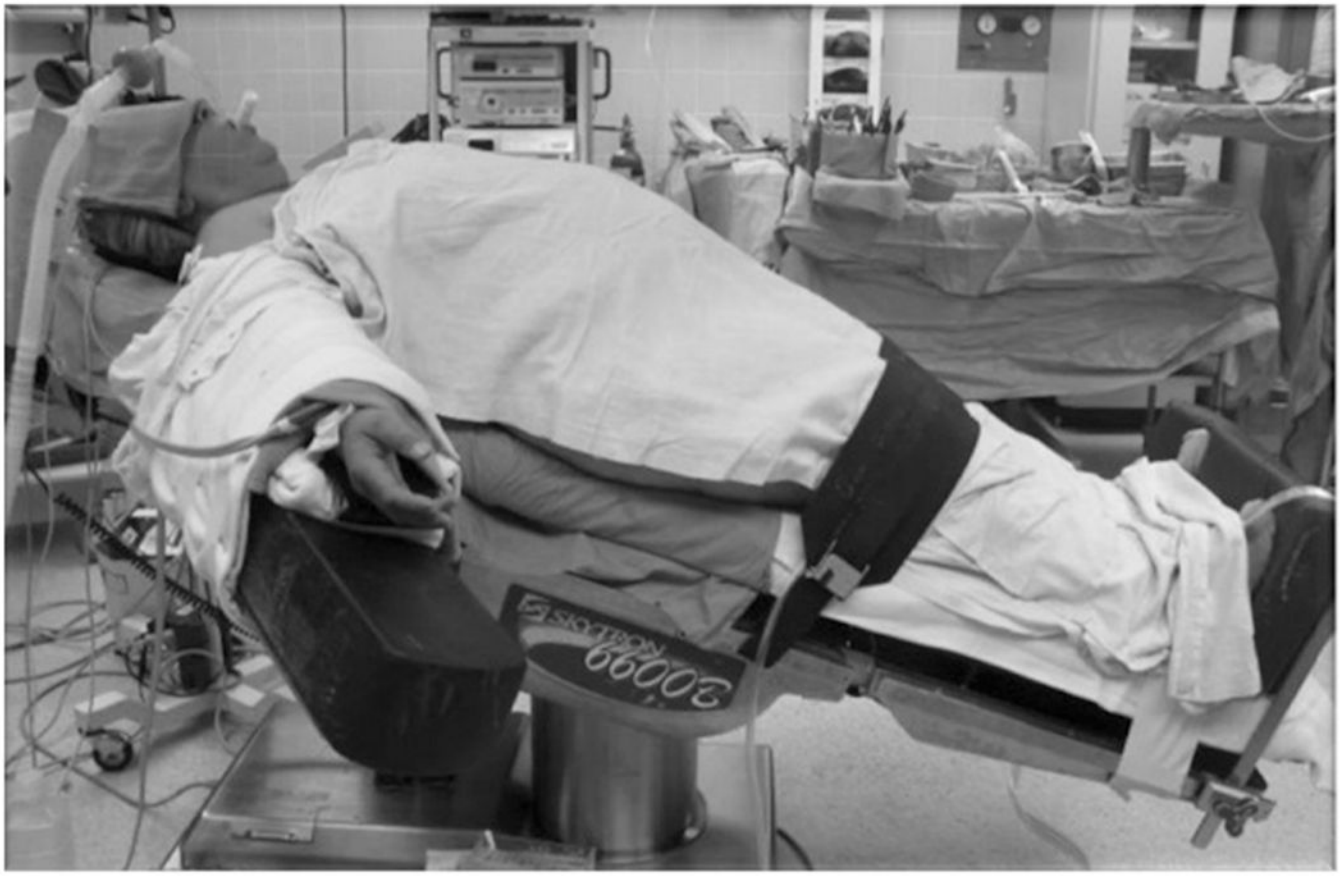

Fig. 1.

Reverse Trendelenburg position with use of head support to facilitate neck flexion and head extension at atlanto-occipital joint. The height of head support (i.e. number of blankets or uncompressible head rest) needed to achieve adequate neck flexion will vary from one patient to another depending on head and neck anatomy and relationship to chest diameter. A good approximation of optimal positioning for laryngoscopy is achieved when an imaginary line can be drawn from the sternal notch to the external auditory meatus. Note foot-board support to prevent the patient from sliding down.

Table 1

Mechanical ventilatory suggestions for volume-controlled ventilation

Predicted body weight (PBW)	Men: PBW (kg) = .9 [height (cm)] – 87
	Women: PBW (kg) = .9 [height (cm)] – 92
Tidal volume (VT)	6–9 mL/kg PBW
Positive end-expiratory pressure (PEEP)	6–12 cm H_2O and titrated to oxygenation.
Recruitment maneuver (RM)	30–40 cm H_2O for 10–40 s if SpO_2 < 90% or reduced by ≥3% from baseline.
	May need to limit if hypovolemic or if blood pressure falls during RM.
Plateau pressure (Pplat)	26–30 cm H_2O
	Larger values may be needed with pneumoperitoneum. Consider esophageal balloon.
Respiratory rate (RR)	Titrated to an $ETCO_2$ of 30–40 mmHg in the absence of other clinical issues. Permissive hypercapnia may be required.
Inspiratory-to-expiratory ratio (I:E)	According to patient's physiology. Monitor for zero flow at end-exhalation.
Fraction of inspired oxygen (FiO_2)	40%–80%, titrated to normoxia.